

## ABSTRACTS OF CARDIOLOGY

**Procaine Amide (Pronestyl in the Treatment of Cardiac Arrhythmias.** J. M. KINSMAN, W. R. HANSEN, and R. L. MCCLENDON. *Amer. J. med. Sci.*, **222**, 365-374, Oct., 1951.

Procaine amide ("pronestyl") was injected intravenously at a rate of 100 mg. a minute and in a total dose of not more than 1 g. in 6 minutes to patients with various types of cardiac arrhythmia. Altogether 41 patients were treated.

In 22 patients with auricular fibrillation no effect was produced with the maximum dose, but in 2 cases of paroxysmal auricular fibrillation this reverted to sinus rhythm after injection of 300 mg. and 550 mg. respectively. There was no response to treatment in 2 patients with auricular flutter. In 2 of 4 patients with ventricular tachycardia the tachycardia stopped promptly; and ventricular extrasystoles were abolished in 12 of 14 patients, although they almost invariably returned in minutes, hours, or days. Subjective toxic effects were few and slight, but the peripheral blood pressure was always lowered, as was also the pulmonary blood pressure. The cardiac output was decreased and the circulation time increased, and there was a prolongation of intraventricular conduction time.

In view of the toxic effects it is recommended that intravenous procaine amide should be used only for patients with paroxysmal arrhythmia in immediate danger of death or when other measures have failed, or for patients under anaesthesia. *C. Bruce Perry*

**The Use of Carboxylic Cation Exchange Resin in the Therapy of Congestive Heart Failure.** A. W. FEINBERG and B. ROSENBERG. *Amer. Heart J.*, **42**, 698-709, Nov., 1951.

Case reports are given of patients treated with a resin mixture which exchanged both potassium and ammonium ions for sodium. By this means it was hoped to avoid the hyperpotassaemia and acidosis produced respectively by the two constituents when employed alone. Five patients with congestive cardiac failure appeared to benefit, the longest period of observation being more than 1 year. The dyspnoea and orthopnoea were relieved in these patients and less frequent use of mercurial diuretics became possible, although venous congestion and oedema were less strikingly improved. In a sixth patient with impaired renal function acidosis developed before his symptoms were relieved, and in another patient gastro-intestinal irritation prevented continuance of treatment. In a patient in the nephrotic stage of chronic glomerulonephritis no diuresis occurred.

The possible toxic effects of these resins are discussed, and the advantages of fewer injections, fewer visits to the clinic, and less rigid dietary sodium restriction are stressed. *Albert Venner*

**Prolonged Cation-exchange Resin Therapy in Congestive Heart Failure.** C. VOYLES and E. S. ORGAIN. *New Engl. J. Med.*, **245**, 808-811, Nov. 22, 1951.

Resodex is a carboxylated cation-exchange resin which is saturated with both ammonium and potassium ions. In the stomach these cations are exchanged for hydrogen ions; in the bowel the hydrogen ions are exchanged for sodium, potassium, and calcium ions. The powdered resin is given in doses of 15 g. 3 times daily in milk or mixed with food. Added calcium is given as calcium lactate, 5 g. daily, or as milk, 16 oz. (450 ml.) daily. Sufficient potassium is supplied in the saturated resin. There is, however, a risk of hypocalcaemia, hypopotassaemia, or acidosis developing; and weekly estimations of the serum calcium, potassium, chloride, and CO<sub>2</sub>-combining power are advisable, as well as the 24-hourly determination of urinary excretion of sodium and chloride.

Three ambulatory patients suffering from chronic congestive failure were given the resin daily for from 5 to 9 months. Before treatment with the resin all were receiving mercurial diuretics twice weekly and were on a diet containing only 500 mg. of sodium. During resin therapy only occasional injections of mercurials were required and 1 patient was able to increase his intake of sodium to 1500 mg. daily. When the resin was discontinued temporarily, congestive failure returned rapidly. During resin therapy the 24-hour urinary excretion of sodium fell to about 15 mg., but the serum sodium, calcium, and CO<sub>2</sub>-combining power showed low to normal values; the serum potassium remained normal. One patient had severe constipation. It is concluded that the administration of a cation-exchange resin is a useful adjunct to the treatment of chronic congestive heart failure. *C. W. C. Bain*

**An Aortic Deformity Simulating Mediastinal Tumour: a Subclinical Form of Coarctation.** C. R. SOUDERS, C. M. PEARSON, and H. D. ADAMS. *Dis. Chest.*, **20**, 35-45, July, 1951.

Three examples of an unusual aortic deformity are described. There were no clinical signs or symptoms. The deformity appeared in the postero-anterior chest radiograph as a mediastinal soft-tissue density overlying a normal aortic arch. Thoracotomy in one case showed that there was a kinking deformity of the aorta toward the pulmonary artery at the attachment of a short ductus arteriosus; minimal narrowing of the aorta was present.

*Kenneth Marsh*

**Vascular Spasm.** G. W. PICKERING. *Lancet*, **2**, 845-850, Nov. 10, 1951.

The idea of severe and localized contraction of an artery, vascular spasm, occurring in the absence of a recognizable

stimulus is entertained too freely and uncritically in current thought and writing. When a large artery such as the femoral is occluded in a normal subject, the distal blood-flow drops to zero for a few minutes and then returns to normal while the occlusion is maintained. Examples are given of transient symptoms of ischæmia following organic arterial occlusion in disease. Many, though not all, of the alleged instances of vascular spasm occurring in limbs, eye, brain, and heart seem to be explicable on other grounds of which the chief is organic arterial occlusion.—[Author's summary.]

**The Cardiovascular Response of Normal Young Adults to Exercise as Determined by the Double Master Two-step Test.** C. B. THOMAS. *Bull. Johns Hopk. Hosp.*, 89, 181-217, Sept., 1951.

The experimental work described in this paper deals with the cardiovascular response to standard exercise in 263 healthy young medical students. The investigation required the use of a modification of the Master test, described as the "double Master two-step test", in which the subjects tested made twice the usual number of trips over the steps in twice the time. Observations of the changes in blood pressure, heart rate, and electrocardiogram (standard limb leads and CF4) were recorded immediately after cessation of exercise.

Great variations in the cardiovascular response were noted, but in general the same subject exhibited similar patterns of response on repetition of the experiment. A group of "hyperreactors" to the exercise test were singled out, and among them obesity, high resting blood pressure, transitory hypertension, high resting heart rate, transitory tachycardia, and a positive cold pressor response were more frequently encountered than among the rest of the subjects. Furthermore, the majority of these subjects had a family history of hypertension with or without coronary artery disease. Thus the concept of an individual constitutional hæmodynamic pattern of response to circulatory stress can be visualized, a pattern which "is as distinctive as the physiological characteristics observed under resting conditions, and which may be of greater significance in terms of future cardiovascular disease."

[This is a well-documented and balanced study on the subject and its publication will undoubtedly stimulate more investigations on similar lines.]

A. I. Suchett-Kaye

**The Mechanism and Significance of the Cold Pressor Response.** H. H. WOLFF. *Quart. J. Med.*, 20, 261-273, July, 1951.

The degree of the rise in blood pressure as a result of immersion of a hand in ice-cold water was originally considered as providing a measure of the greater reactivity in this respect of subjects with established or potential essential hypertension, since the rise in blood pressure was found to be greater than in healthy subjects. This claim has not since been substantiated.

The present investigation was undertaken in order to clarify what determined the height of the cold pressor response and what significance, if any, can be attached to it. The pain experienced during the test was already recognized as an important factor to account for the response, but the question had to be clarified whether the afferent impulses responsible for the rise in blood pressure excited a vasomotor reflex at some level below that at which pain is perceived, or whether the perception of pain was a pre-requisite for the increase in blood pressure. Tests carried out in 3 patients with hemianæsthesia due to cerebral vascular lesions, 6 with hysterical anæsthesia, 2 with analgesia due to syringomyelia, and one with a peripheral-nerve lesion demonstrated that immersion of the anæsthetic limb did not cause any significant increase in blood pressure.

The importance of avoiding any rise in blood pressure due to emotional factors, such as apprehension in connexion with the test, is stressed. It is concluded that the vasoconstriction underlying the cold pressor response occurs only as a reaction to the pain experienced during the test and is not the result of a reflex. This test therefore provides a measure of an individual's sensitivity to pain, and its value lies in further research on this subject rather than in connexion with hypertension.

A. Schott

**The Use of Quinidine in Established Auricular Fibrillation and Flutter.** D. HOLZMAN and M. G. BROWN. *Amer. J. med. Sci.*, 222, 644-652, Dec., 1951.

Of 57 patients with auricular fibrillation or flutter it was possible to establish normal sinus rhythm in 30, but in 14 this could be maintained only during a follow-up period of 6 months. It appears to the authors that long-standing arrhythmia, cardiac enlargement or rheumatic heart disease does not satisfactorily respond to quinidine, but that when such a response is obtained it is usually in patients who have been given moderate doses.

The dosage of quinidine used was 0.2 g. every 2 hours for 7 doses. If normal sinus rhythm was established a maintenance dose of 0.2 g. was given 6-hourly for a month and then discontinued.

James W. Brown